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LETTERS TO THE EDITOR

Coronary Thrombosis and Myocardial Infarction

Muller (1), in a selective historical review, has concluded that the primacy of occlusive coronary thrombosis in the genesis of transmural myocardial infarction is now a settled matter. But is it?

Reports of early angiographic, thrombolytic and emergency surgical interventions in acute ischemic heart disease have established the presence of intraluminal clots or thrombi in the initial stages of myocardial infarction that diminish in frequency over the following hours as the infarct evolves. How can these observations be reconciled with the large body of data from necropsy studies showing a generally lower incidence of occlusive thrombi in the first day or two following infarction and an increasing yield of thrombotic occlusions in patients dying several days or more after onset of infarction? Detailed studies of "classical" occlusive coronary thrombi have shown a high frequency of associated complications such as intimal hemorrhage and plaque rupture. The thrombus itself is commonly an intimate admixture of fibrin, platelets, atheromatous debris and intramural hematoma. This is what pathologists see in fatal myocardial infarction, but may not be what clinicians visualize in vivo during early acute myocardial infarction.

It is proposed that occlusive coronary thrombosis may be, in many instances at least, a biphasic phenomenon. Given the morphologic background of major obstructive coronary atherosclerosis, dynamic vascular factors such as spasm and/or transient platelet aggregation may induce a thrombus at a point of narrowing. These unstable clots probably consist largely of platelets and a loose fibrin meshwork. Depending on associated factors, such thrombi may play a greater or lesser role in the development of irreversible ischemic injury in the territory of supply of the involved vessel. The initial clot, because it is part of a dynamic process, will often lyse spontaneously or with the help of intracoronary streptokinase, although occasionally it may persist and evolve into a fixed thrombus. If an infarct becomes established, stretching of the akinetic area, exudation, capillary endothelial swelling and secondary stasis thrombi in the microcirculation of the necrotic zone (which, in fact, do occur) lead to cessation of the intramural circulation. Resultant redistribution of coronary flow and altered pressure gradients in the epicardial arteries, possibly in association with recurrent spasm and platelet-induced endothelial injury, may produce disruptive plaque events and a permanent occlusive thrombus at the site of major stenosis. It is not necessary to postulate propagation or continuity of thrombi from the small vessels of the infarcted zone back to the point of proximal stenosis and occlusion.

Baroldi (2) has again given a reasoned assessment of the role of coronary thrombosis in the pathogenesis of myocardial infarction and concluded that intimal hemorrhage, rupture of atheroma and occlusive thrombi are epiphenomena of increased peripheral resistance and/or mechanical effects (spasm, wall motion secondary to exerted contractility) on the atherosclerotic plaque. Roberts (3), Silver (4) and colleagues and Olsen (5) support this interpretation, and the latter also suggests a possible two-stage sequence in the development of occlusive coronary thrombi.

The juxtaposition of Gorlin's paper (6) to that of Muller (1) is most appropriate. Dynamic vascular factors, including evanescent platelet aggregation thrombi, most likely play a greater part in the genesis of myocardial ischemia and infarction than do fixed or permanent thrombi which are usually secondary events. Clearly, there is room for further evolution of our conception of coronary thrombosis vis à vis myocardial infarction and of the pathophysiologic mechanisms involved.

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3 Roberts WC Coronary thrombosis and fatal myocardial ischemia. Circulation 1974;49 1–3

4 Silver MD, Baroldi G, Mariani F The relationship between acute occlusive thrombi and myocardial infarction studied in 100 consecutive patients Circulation 1980;61 219–27

5 Olsen EGJ Ischemic diseases of the myocardium and its complications. In Ref 2 393–439

6 Gorlin R Dynamic vascular factors in the genesis of myocardial ischemia J Am Coll Cardiol 1983;1 897–906

Reply

My thoughts on the role of thrombosis in myocardial infarction are not as far from those of Heggtveit as his comments imply. We agree that thrombosis is generally present in cases of transmural infarction and that there are unanswered questions "regarding the cause of the thrombosis itself" as I stated in the article. Heggtveit's proposed mechanism of thrombus formation is a contribution to the continuing inquiry about the etiology of the thrombus.

We may differ in our assessment of the harm the thrombus itself may cause and the potential importance of thrombolysis. Regardless of the cause of the thrombus, I believe there is a stage in the development of myocardial infarction in which coronary flow to potentially salvageable myocardium is impeded by clot. We have available the means to lyse such a clot and should devote considerable energy to determining if thrombolysis is beneficial, in parallel with the efforts advocated by Heggtveit to deepen our understanding of the mechanism causing thrombosis.

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